in patients who have received preoperative α-adrenergic blockade.[20]

The intraoperative responses in our patient were similar to those of hypertensive patients with pheochromocytoma. Some lability of blood pressure and heart rate was seen, especially upon manipulation of the tumor, and treated with phentolamine. Several episodes of nodal and ventricular tachycardia in association with the hypertensive responses to manipulation of the tumor resolved after control of the blood pressure. Hemodynamic monitoring showed that, before anesthesia, systemic vascular resistance was below the normal range, cardiac index was increased and blood pressure was normal. These findings are consistent with the hypothesis that the patient had a hormonal milieu blunting the usual α-adrenergic response to norepinephrine. Nevertheless, during manipulation of the tumor, both blood pressure and vascular resistance increased considerably, suggesting that under these circumstances the catecholamine balance changed to an α-adrenergic predominance. Subsequent to clamping the venous drainage from the tumor, vascular resistance and blood pressure fell.

The spectrum of the presentation of pheochromocytoma continues to expand. Hypertension may be absent despite excess norepinephrine secretion. Fever of unknown origin, anemia, extreme elevation of the erythrocyte sedimentation rate and hepatopathy may be added to the spectrum of paraneoplastic syndromes caused by this neoplasm.

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Remembrance of Things Past: Aortobronchial Fistula 15 Years After Thoracic Aortic Homograft

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THE EVOLUTION of prosthetic vascular grafts has included the development of diverse materials, starting with the Vinyon-N fabric of Blakemore and Voorhees and progressing to the use of Dacron, expanded Teflon, bovine carotid artery and preserved human umbilical vein. In the 1950s intense interest attended the use of human cadaver arteries, preserved in various ways, for vascular substitutes. These arterial homografts were readily available and technically easy to handle, had a reasonable patency rate in the short term and were used commonly in many different clinical circumstances. The first replacements of the aorta for aneurysm and occlusive disease used these conduits. About 1956, however, it became clear that the homograft preservation process and constant pulsatile arterial pressure led inevitably to a loss of structural integrity and graft degeneration.[1,2] Aneurysm formation or fibrosis leading to graft rupture or occlusion caused the use of these grafts to be abandoned, a move hastened by the parallel development of acceptable polyester grafts. Arterial homografts were still available in many hospital operating rooms until the mid-1960s but subsequently have almost completely disappeared from use. Homograft tissue is now rarely used in cardiovascular surgical procedures, except for cardiac valve replacement using tissue-bearing prostheses.[3,4]

We describe the case of a patient who underwent aortic repair using an arterial homograft, in whom lethal complications developed 15 years later because of this graft. With this report we hope to alert clinicians to the potentially catastrophic complications that may result from these grafts and we propose diagnostic maneuvers that may forestall them.

Report of a Case

In 1965 a 20-year-old man sustained a blunt thoracic deceleration injury in an auto accident. He underwent

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placement of a Teflon interposition graft to repair a tear of the descending thoracic aorta. This procedure was complicated by a graft infection that required replacement of the Teflon graft with an aortic homograft. Subsequently the patient did well.

Fifteen years later the patient, now 36 years old, was seen by his private physician because of worsening of chronic asthma associated with atopy. He had no evidence of infection and no history of hemoptysis, but he had recently increased his cigarette consumption. Roentgenograms of the chest confirmed the previously noted findings of chronic left upper lobe fibrosis and aortic calcification (Figure 1). Oral aminophylline therapy was begun, resulting in lessened wheezing and improved pulmonary function, but the patient had ongoing malaise. Three weeks later he coughed up a large amount of blood and shortly thereafter had respiratory arrest. Seattle Medic-I[5] paramedic personnel found the patient unresponsive and apneic and with a pulse rate of 40. An orotracheal tube was inserted and he was given 100% oxygen; large-bore intravenous lines were placed and the patient was transported to the emergency ward of Harborview Medical Center under continuous cardiopulmonary resuscitation.

On arrival, the patient was arousable. He had a pulse of 115 per minute and a systolic blood pressure of 100 torr (mm of mercury). Blood was noted in the endotracheal tube, and no breath sounds were heard over the left upper thorax. Hoarse rhonchi were audible throughout the chest. A chest roentgenogram showed a heavily calcified aortic arch and descending thoracic aorta; the left upper lung field was completely opacified. Arterial blood gas determinations done while the patient was receiving 100% oxygen showed a pH of 7.02, a partial carbon dioxide pressure (P_{CO_2}) of 69 and a partial oxygen pressure (P_{O_2}) of 217 mm of mercury. Hematocrit was 35%.

The patient was taken directly to the operating room with the intent of doing bronchoscopy and thoracotomy, with a presumptive diagnosis of a ruptured pseudoaneurysm of the descending thoracic aorta. On arrival in the operating room he again had massive hemoptysis; his endotracheal tube was cleared, then advanced into the right mainstem bronchus. Despite these maneuvers, the patient had respiratory, followed by cardiac, arrest, and cardiopulmonary resuscitation was reinstituted. An emergency left anterior thoracotomy was done. Entry into the chest was remarkably difficult because of extensive fibrotic adhesions and massive bleeding. He was found to have an extremely brittle, calcified pseudoaneurysm of the aortic arch from the left subclavian orifice to the midthoracic aorta; this had ruptured into the left upper lobe of the lung. The patient died before the hemorrhage was controlled.

Postmortem examination showed dense periaortic adhesions in the left thorax; multiple pseudoaneurysmal dilations of a patent, fibrotic aortic homograft conduit just distal to the left subclavian artery were noted. One of these aneurysms had ruptured into the left upper lobe bronchus (Figure 2).

Comments

The occasional but disastrous development of an aortoenteric fistula, pseudoaneurysm or prosthetic vas-

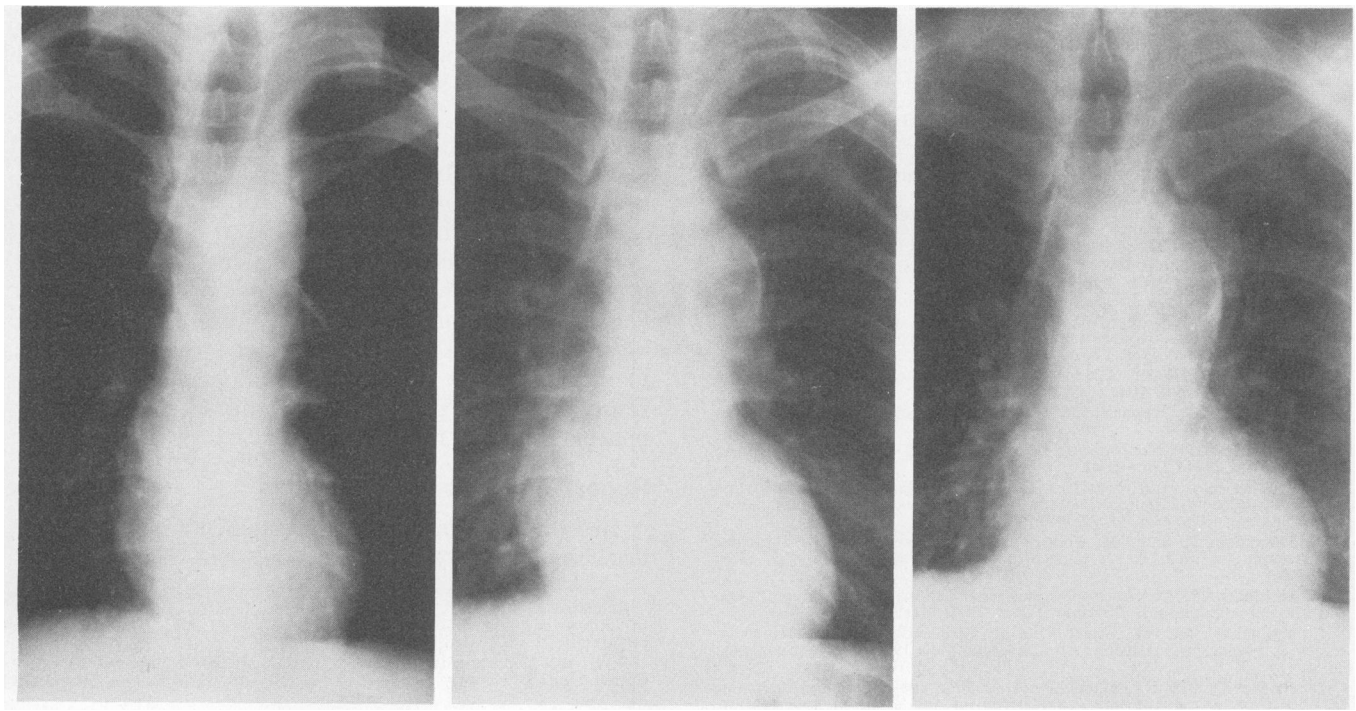

Figure 1.—Chest roentgenograms five months (left), ten years (center) and 15 years (right) after thoracic aortic interpositional graft with cadaver arterial homograft. Note development of aortic homograft calcification.

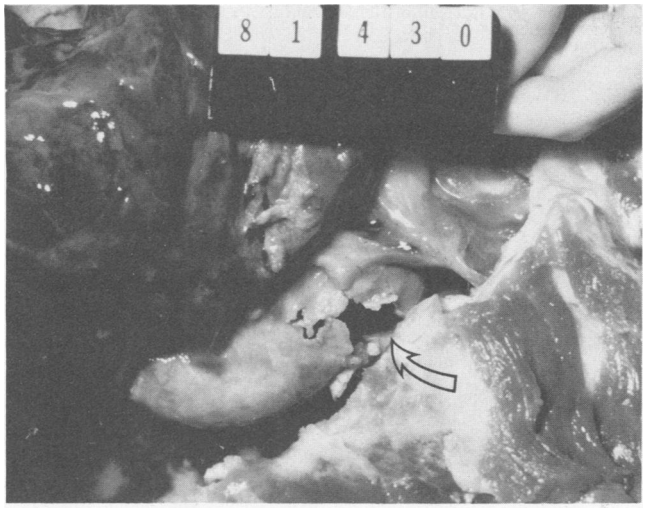

Figure 2.—Photograph at postmortem examination showing ruptured pseudoaneurysm of thoracic aortic arterial homograft **(arrow).** The pseudoaneurysm ruptured into the left upper lobe of the lung **(upper left corner of photograph).** (Photograph courtesy of King County Medical Examiner's Office.)

cular graft infection reminds cardiovascular surgeons that the implantation of foreign materials into the arterial tree has an ongoing and cumulative risk of complication. The Proustian title of our report underscores the fact that such complications may be significantly delayed—in this case for a decade and a half.

The use of homograft tissue for vascular replacement has definite appeal, for such conduits are readily available and material qualities such as elasticity, compliance and strength would seem to be relatively advantageous. Unfortunately, the use of fresh cadaver material for vessel or valve grafts generally violates the "self/not-self" immune barrier,[6] and such material is eventually rejected by the host. Similarly, though various preservation techniques—such as freeze-drying,[7] immersion in ethylene oxide[8] or β-propiolactone,[9] or electron irradiation,[7]—appear to blunt or ameliorate the antigenic stimulus, the benefits of such methods are apparently offset by the production of structural damage to the graft at the cellular level, and these tissues thus generally undergo necrotic or fibrotic degeneration. Initially, arterial homografts generated enthusiasm because of excellent short-term results when used for arterial bypass or replacement.[10-12] Within a year or two after implantation, however, these grafts developed unacceptably high rates—greater than 50%—of thrombosis, aneurysm formation or infection[1,2,13-15] and their use was rapidly eschewed in favor of innovations in plastic grafts. Homograft tissue is now rarely used in cardiovascular operations—occasionally for conduits in the Fontan procedure and for cardiac valves,[3,4]—where it has been associated with modest short- and medium-term results.

Massive hemoptysis, a common occurrence during the tuberculosis era, is now seen much less often and usually arises from blunt or penetrating chest trauma or malignancy involving the bronchial arterial circulation.[16] Hallmarks of management include simultaneous resuscitation and diagnosis, the latter by means of rigid bronchoscopy, chest roentgenography and pulmonary and bronchial arteriography. Massive persistent bleeding may be controlled immediately by intubation of the contralateral (normal) bronchus by single- or double-lumen endotracheal tubes or Fogarty catheters,[17] by angiographic embolization[18] or by emergency thoracotomy and lobectomy or pneumonectomy.[19] The death rate remains high.

In our patient a lethal outcome was virtually preordained because the diagnosis was not suspected until his homograft pseudoaneurysm ruptured into the bronchial tree. A thoracotomy to resuscitate the moribund patient met impenetrable adhesions and massive bleeding in the left upper hemithorax. Balloon tamponade of the thoracic aorta[20] proximal to this point via the axillary artery might have been considered but would have risked carotid occlusion and stroke. Had the presence of incipient graft rupture been anticipated, or even considered, when the patient presented three weeks earlier with worsening of his asthma, elective thoracic aortic replacement (probably with en bloc left upper pulmonary lobectomy) using adjunctive cardiopulmonary bypass might have offered him the chance of long-term survival.

The vast majority of patients who underwent placement of preserved homograft arteries for bypass or interposition in the 1950s were elderly persons who had arteriosclerosis, and it is unlikely that many have survived the subsequent quarter century. A number of infants and children, however, underwent homograft arterial interposition grafting for various congenital cardiovascular defects during that time. Robert Gross, MD, inserted homograft aortic conduits in almost 100 young patients who had aortic coarctation, for example (reported in conversation, 1981). These persons have now reached young adulthood. The literature shows, with few exceptions,[14,15] little long-term follow-up of these or other young patients who had implantation of these cadaver arterial grafts. We suspect that, as in our case, the obvious but chronic chest roentgenographic abnormalities, coupled with a lack of appreciation of the well-documented but obscure natural history of arterial homografts, may prevent recognition of the lethal potential of these grafts.

Our case's fatal outcome, combined with the known cumulative incidence of complications of these grafts, leads us to suggest that all patients, asymptomatic or not, who have had placement of such homograft vascular conduits at any time in the past should be considered for elective graft replacement. At the least, such persons should be serially evaluated by computed tomographic scan or ultrasound studies, if not arteriography, to delineate possible evidence of graft degeneration—dilatation, aneurysm formation, extensive calcification and so on. Such findings might lead to a decision favoring elective graft excision and replacement. Thus, we are in agreement with Meade and col-

leagues, who closed a 1966 report on the long-term fate of arterial homografts by commenting: "Confirmed follow-up of all . . . cases is essential since graft failure may occur at any time regardless of how long the graft has been placed."[13(p399)]

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